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PESTS NOT KNOWN TO OCCUR IN THE UNITED STATES OR OF LIMITED
DISTRIBUTION NO. 90: Xanthomonas campestris pv. oryzicola

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20782

Disease

RICE BACTERIAL LEAF STREAK

Pathogen

Xanthomonas campestris pv. oryzicola (Fang, Ren, Chen, Chu,
Faan & Wu 1957) Dye 1978

Other Names

Xanthomonas oryzicola (Fang et al. 1957)

Xanthomonas translucens (Jones, Johnson & Reddy 1917) Dowson
1939 f. sp. oryzicola (Fang et al. 1957) Bradbury 1971

Xanthomonas translucens (Jones et al. 1917) f. sp. oryzae
(Uyeda & Ishiyama 1928) Pordesimo 1958 has been incorrectly
used.

Class:

Schizomycetes:

Order: Family

Pseudomonadales: Pseudomonadaceae

Economic
Importance

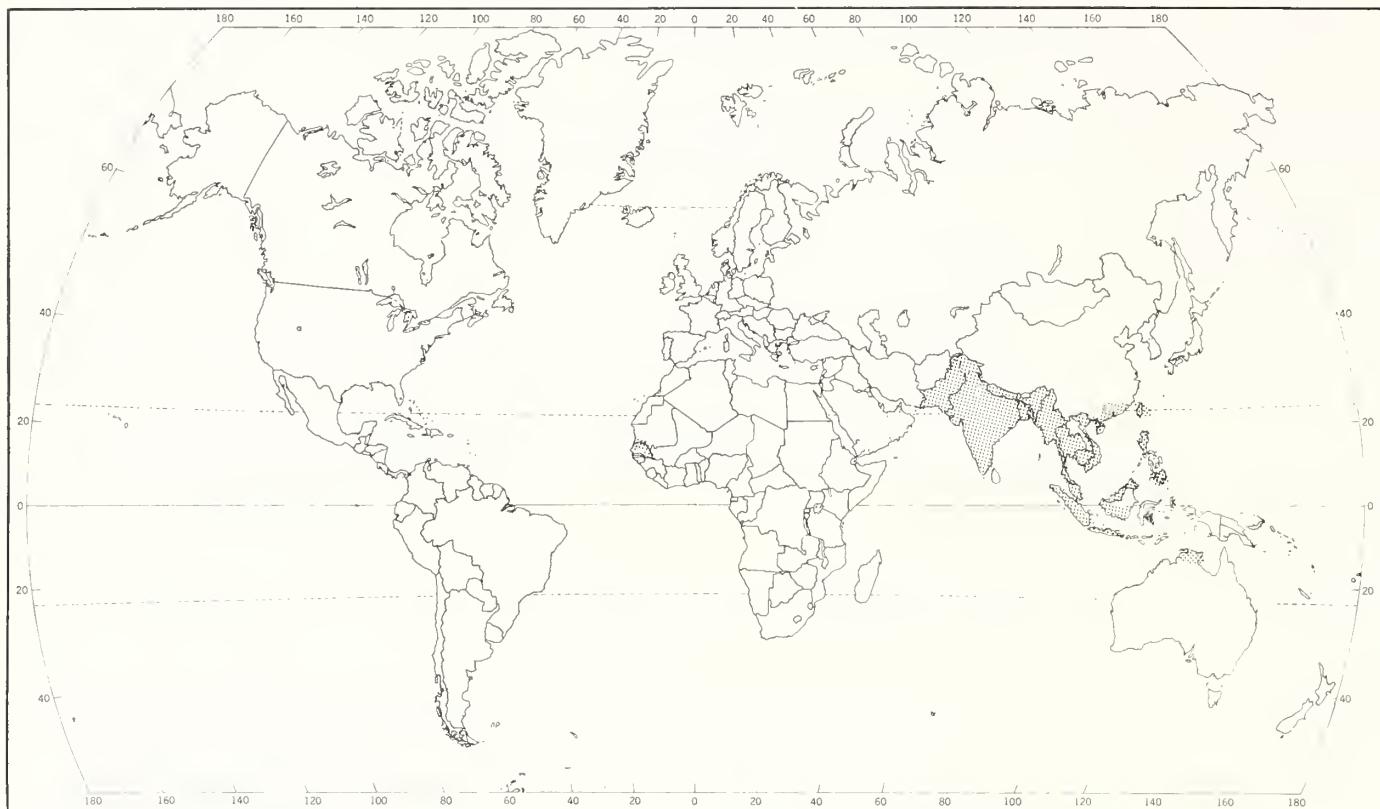
Rice bacterial leaf streak is the second most serious bacterial disease of rice. Wide use of short-term, nitrogen-responsive cultivars and a high rate of nitrogen application, combined with favorable weather, have greatly increased the disease (Ho 1975). Losses in central India ranged 5 to 30 percent depending upon environmental factors and the cultivar (Chand et al. 1972, Naik et al. 1973); in the northern area, disease intensity affecting 80 percent of leaf area resulted in 61 percent yield loss (Singh et al. 1980). Elsewhere, no losses were considered significant. In Malaysia, although the disease was severe especially between active tillering stage and young ear formation stage, the disease did not significantly affect yield (Ho 1975). In the Philippines, no losses were significant in either the wet or dry seasons (Opina and Exconde 1971).

Hosts

Apparently restricted to Poaceae on one major crop, Oryza sativa, rice. Another naturally infected host includes O. perennis, a perennial wild rice (Reddy and Nayak 1975). Tests of 49 wild Oryza species and strains indicated possible alternative hosts (Ou 1985).

General
Distribution

Generally distributed in the southeastern Asian tropics. It has been reported in several West African countries (Srivastava (pers. comm.) from Ou 1985). The following countries were listed by the Commonwealth Mycological Institute (1982) unless cited otherwise: Australia (Northern Territory), Bangladesh, Burma (Khush 1977), Cambodia, China (Guangdong including Hainan



Xanthomonas campestris pv. oryzicola distribution map.

Dao, Khush (1977) reported Taiwan), India, Indonesia (Java, South Sulawesi (Tominaga et al. 1978), Sumatra and South Borneo (S. H. Ou, pers. comm.)), Malaysia (Malaya and Sabah; Sarawak (S. H. Ou, pers. comm.)), Nepal (European and Mediterranean Plant Protection Organization 1977), Pakistan (Khush 1977), Philippines, Senegal (Trinh 1980), Thailand, and Vietnam.

Bacterial leaf streak reported in the United States (Devadath 1984) based on a Xanthomonas find on eastern wildrice (Zizania aquatica) (Kernkamp et al. 1976) is incited by another bacterium. X. campestris pv. oryzae is not known to infect Zizania and has not been detected in the area on rice (O. sativa).

Characters

Obligately aerobic, oxidative, Gram-negative, nonspore-forming, motile, capsulate, straight 0.4-0.6 X 1.0-2.5 μm rods with single polar flagellum (Bradbury 1970b, 1984).

Colonies on nutrient agar smooth, opaque, glistening, circular, convex, entire, whitish then straw to pale yellow, diameter about 1 mm in 3 days (Goto 1964 from Bradbury 1970b). Pigment

characteristically brominated aryl polyene; colony butyrous or viscid, mucoid on nutrient agar with 5 percent glucose or sucrose (Bradbury 1984, Starr 1981). Temperature thresholds: minimum 8 °C, optimum 25-28 °C, maximum 38 °C, thermal death point 51 °C (Bradbury 1970b).

This bacterium may be confused with X. campestris pv. oryzae (Ishiyama) Dye, but their symptoms on rice (See Characteristic Damage) distinguish them easily. In culture, X. campestris pv. oryzae grows slower on nutrient agar (colony diameter 1-2 mm in 5-7 days), fails to peptonize litmus milk, rapidly hydrolyzes cellulose, requires organic nitrogen for growth (Goto 1964 from Bradbury 1970a), and fails to hydrolyze starch (Bradbury 1970a). Cruz et al. (1984) differentiated them on four phenotypic tests. Suetsugu et al. (1983) found isolation with selective medium, confirmation with antiserum, and observation of parenchyma tissues of the infected leaves were useful in detecting X. campestris pv. oryzicola. Reddy (1981) used immunoelectrophoresis to differentiate the two pathovars.

Characteristic Damage

Rice bacterial leaf streak is a foliar disease. At first, stripes (Fig. 1) are 0.5-1 mm X 3-5 mm, interveinal with margins linear (not wavy) (Bradbury 1970b, European and Mediterranean Plant Protection Organization 1977, Ou 1985). Streaks are a watery, translucent dark green, sometimes ringed with yellow on susceptible cultivars (Tominaga et al. 1978, Ou 1985). Streaks later turn translucent yellow to yellow-orange (Ho 1973, Singh 1969), brown, and then grayish white because of saprophytic growth (Ou 1985). Streaks may coalesce along the leaf length and cover the entire surface, resulting in their blighted, ragged appearance (Devadath 1984, Singh 1969, Tominaga et al. 1978).

Soon after lesions develop, these streaked portions exude tiny yellow or amber bacterial droplets (Fig. 1) (Bradbury 1970b, Ho 1973). The droplets dry to tiny beads with or without cirrhus-like stalks, or to large diffused, irregular shapes and cirrhi depending upon moisture conditions. The delicate cirrhuslike exudates may be blown away by a dry wind, but the beadlike exudates stick firmly to the leaf surface, protected from wind dispersal (Shekhawat and Srivastava 1972b), and later fall into field water (Faan and Wu 1965, Ou 1985).

The disease more severely affects younger than older growth stages and younger leaves more than older leaves at any stage. Severity increases during vegetative rice growth until panicle initiation stage and gradually declines into the booting stage. By the milk ripe stage, the plant recovers (Ho 1973, 1975, Rao and Devadath 1977b, Shekhawat and Srivastava 1972b).

(Fig. 1)



Rice bacterial leaf streak from initial symptom to coalition of lesions with bacterial ooze on watersoaked lesions (Courtesy T. W. Mew, International Rice Research Institute).

Besides varying with host age, severity varies with the rice cultivar and the bacterial isolate, affecting the amount of exudate produced (Shekhawat and Srivastava 1972a, Shekhawat et al. 1972) and the lesion length. Lesion length ranges from less than 0.5 cm to 5-10 cm (Ou 1985). Without mechanical injury, hairy cultivars may be more prone than glabrous ones to infection (Rao and Devadath 1979b).

The diseases caused by the two Xanthomonas pathogens to rice are easily differentiated by early symptoms. Rice bacterial leaf streak is characterized initially by fine translucent streaks between leaf veins. Rice bacterial leaf blight (incited by X. campestris pv. oryzae) initially appears near upper leaf margins as watersoaked stripes, which enlarge and coalesce into yellow lesions with wavy edges (Fig. 2); if the leaf is injured, symptoms may appear in leaf areas other than in the

margins. Streak lesions exude amber or yellow bacterial droplets, but blight lesions exude milky or opaque droplets. The blight may produce two other symptoms: (1) kresek, in which leaves and then plants wither, or (2) young leaves that are pale yellow on mature plants (Bradbury 1970a, Ou 1985).

Streak appears only on leaves in parenchymatous tissue. Blight affects the vascular system of leaf and stem (Fang et al. 1957, Goto 1964, Shekhawat and Srivastava 1972b). With blight, glumes of young and green grain show discolored spots with a water-soaked margin, later turning gray or yellowish white (Ou 1985).

(Fig. 2)



Rice bacterial leaf blight (Courtesy T. W. Mew, International Rice Research Institute).

Detection
Notes

Movement of contaminated rice straw, plant debris, or seed from infected plants can introduce this pest into new areas. Rice straw and other rice material, unrotted or partly so, harbors this bacterium for a long period. The bacterium remained infective for 2 years in diseased leaves stored at about 20-31 °C (Devadath and Dath 1970). Seed also presents a high risk with about 10 percent seed transmission and no visible indication that seed is infected (Shekhawat and Srivastava 1972a).

To prevent introduction of rice pathogens and insects into the United States, Title 7 of the Code of Federal Regulations regulates the entry of rice and its products. Rice straw, hulls, and chaff in packing materials are prohibited by Part 319.69. Rice straw imported for commercial use is permitted entry by Part 319.55 with a USDA permit subject to inspection and treatment. Propagative materials including seed of Oryza spp. enter only for scientific purposes by Departmental permit under Parts 319.37 and 319.55.

Survey by looking at young leaves for interveinal translucent streaks. Examine young lesions for tiny yellow beads.

Submit for identification, suspect plant material dried, pressed, and labeled in sealed double containers (one inside the other) with screw tops.

Biology and
Etiology

The pathogen may be carried from season to season in seed, infected plant debris, soil, or water. Alternative hosts, such as O. perennis (Reddy and Nayak 1975), may also play a role. Infected volunteer rice plants provide inoculum, especially for double-cropped rice areas (Devadath 1984).

Infected seed is a good potential inoculum source. The bacterium can survive up to 5 months in seeds stored at 15-30 °C. Seed transmission occurs within 15 days of planting under protracted high humidity with frequent rains, but a dry spell indefinitely delays or completely arrests transmission (Devadath 1984). Infection begins with the pathogen (probably below the glumes) contaminating the developing plumule, successively infecting the coleoptile, leaf sheaths, and leaves. The first leaf then carries the pathogen to aerial parts (Shekhawat and Srivastava 1972c).

Infected leaf debris could provide an inoculum source. The bacterium remains viable in pieces of unrotted leaves. Infectivity lasted 10 months in leaves mixed with soil (whether leaves were buried or left on the surface, or whether soil was sterilized or not). Survival appeared to depend on moisture, temperature, and incubation period (Devadath and Dath 1970).

Without leaf debris, contaminated soil can also serve as an inoculum source for a few months. Bacteria inoculated onto unsterilized soil and onto sterilized soil remained infective for 7 and 18 weeks, respectively, at 15-30 °C (Devadath and Dath 1970). Soil type, antagonistic microorganisms present, and physico-chemical properties of the soil affected longevity (Devadath 1984).

During the dry season, contaminated bodies of water may harbor the bacterium and provide inoculum in the new cropping season. The bacterium lives up to 90 days in water at 15-20 °C (60 days at 25-45 °C). Antagonistic microorganisms present and the chemical nature of the field water may also determine the survival period (Devadath 1984). Used for irrigation, this water may introduce the pathogen into rice fields and carry it from field to field (Devadath 1984, Faan and Wu 1965). Contact of rice leaves with contaminated field water is essential to infection as the pathogen cannot move from water to leaf through the leaf sheath (Rao and Devadath 1977a).

In whatever manner the bacterium survives during noncropping seasons, infections are incited by the bacterium penetrating leaves through stomata or wounds (Devadath 1984, Ou 1985). It then multiplies in the substomatal cavity, progresses intercellularly in the parenchyma, multiplies in large masses in air spaces near the midrib, and oozes through the stomata onto the leaf surface (Shekhawat and Srivastava 1972b) under moist conditions during the night (Ou 1985). This exudate is the inoculum for secondary spread.

Moistened by dew or rain, the bacterial cells in the exudate are dispersed and spread by wind to cause new infections on the same leaf or on other leaves. Rapid spread occurs throughout an entire field, especially after such wind-driven rains as rainstorms or typhoons. Infection is primarily restricted to exposed leaves during typhoons, and secondary spread only occurs when there is rain and wind. Thus bacterial leaf streak apparently disappears almost as rapidly as it appears because the canopy of new leaves covers old infected leaves (Ou 1985).

Successful infection depends on continuous high humidity (relative humidity 74 percent or more) or dew for 2-3 mornings. No infection occurs irrespective of temperature if humidity is less than 50 percent for 15 days after inoculation. With mean humidity between 41.0 to 59.0 percent, infection occurs with prolonged incubation under cool, humid nights and dew covering leaves during early morning. A short dry spell may interrupt successive infection of young leaves, and thus, break the disease cycle (Shekhawat and Srivastava 1972a).

After infection, the critical factor is temperature, regardless of humidity, in disease development. A range of 26.0 to 32.0 °C favors lesion development while temperatures below 22.4 °C retards it (Devadath 1984, Shekhawat and Srivastava 1972a).

Wind and windblown dust aid dispersal and invasion (Devadath and Dath 1970, Hashioka 1969). Rainy days, high humidity, optimum temperature range, and overcast days favor disease spread (Rao and Devadath 1979a). These factors combined with heavy nitrogenous fertilization and susceptible plant age, result in rapid disease spread (Chand et al. 1972, Devadath 1984, Ho 1975).

Leafhoppers, grasshoppers, man, and agricultural equipment can mechanically transmit the bacterium (Devadath 1984, Rao and Devadath 1977a). The disease is also associated with leaf-rollers, leaffolders, and hispa, since the bacterium readily enters insect-damaged tissue (European and Mediterranean Plant Protection Organization 1977).

Controls

Practices to reduce the amount of inoculum in the early, more susceptible rice stages included plowing under infected crop residue and volunteer rice plants, inundating the field for several days 2 months before sowing or transplanting, and eradicating susceptible wild rice species in and around rice fields (Devadath 1984) before the first crop and between successive rice crops. Inoculum could also be reduced by planting seed from uninfected plants. Chemical seed treatment (Shekhawat and Srivastava 1971), and seed treatment followed by hot water treatment reduced seed transmission (Shekhawat et al. 1969). Also planting resistant cultivars (Devadath 1984, Goto 1965, Ou 1985, Shekhawat et al. 1972) could reduce severity.

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